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SUCCESSFUL TREATMENT OF A SOLITARY SKULL METASTASIS IN A CHILD WITH WILMS' TUMOR

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Abstract

This report presents the successful treatment of a child with a solitary metastatic lesion to the calvarium following treatment for Stage III anaplastic Wilms' Tumor.

Keywords

Wilms' Tumor; recurrence; metastatic; nephroblastoma; skull metastasis

1. INTRODUCTION

Overall survival for children with favorable histology Wilms' Tumor (WT) is greater than 90% at five years. Combined data from the National Wilms' Tumor Study (NWTS) groups I, II, and III was reviewed by Breslow, et al. and showed a 5-year OS of 66% for patients with both favorable and unfavorable histology Stage IV disease at the time of diagnosis.[1] For those presenting without distant metastasis at diagnosis, but who recurred with metastatic disease at some point following treatment, the 5-year OS is only 40%.[1] Bony metastatic disease in WT is uncommon; bony disease without distant metastasis elsewhere is exceedingly rare.[2] Here, we present a case of a solitary bony metastatic lesion in the calvarium, which presented after completion of the treatment course in a child with WT.

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Conflicts of interest: none.

2. CASE REPORT

2.1

An 18-month old girl presented with an abdominal mass on routine well-child visit. A CT scan showed a large intraabdominal mass appearing to arise from the left kidney. At laparotomy, a 15 × 11 × 6 cm mass was encountered, which was found to be arising from the left kidney and compressing the stomach, the pancreas, and the spleen. A small amount of tumor rupture occurred while dissecting the tumor free from the left hemidiaphragm, to which it was adherent. A left radical nephrectomy was performed, along with resection of as much involved diaphragm as was feasible to allow primary repair. Pathology revealed a blastemal-predominant WT with multiple foci of anaplasia with positive surgical margins at the diaphragm. Diagnosed with Stage III WT with diffuse anaplasia, she was treated with a combination of vincristine, cyclophosphamide, doxorubicin, and etoposide for 24 weeks per Regimen I of the National Wilms Tumor Study V, along with 1080cGY external beam radiation to the left flank and left lung base/hemidiaphragm.

2.2

Follow-up imaging of chest and abdomen at completion of therapy showed no signs of metastatic disease, and she was scheduled to have her implanted venous access device removed. During her routine pre-operative visit, she still had alopecia and was noted to have a focal area of painless, soft-tissue swelling overlying the vertex of her skull. Cross-sectional imaging of the head showed lytic destruction of the bone with a lesion extending into the subgaleal soft tissue as well as into the epidural space without involvement of the cerebrum (Fig. 1). Technetium-99 pyrophosphate scan showed increased uptake in the posterior skull with no additional sites of bony recurrence. An incisional biopsy was performed which confirmed WT with diffuse anaplasia. She underwent two cycles of intensive chemotherapy with ifosfamide, carboplatin, and etoposide. She then received high dose chemotherapy with thiotepa and cyclophosphamide conditioning, followed by autologous stem cell rescue. Finally, she received 3,060 cGY external-beam radiation to the affected region of the skull.

2.3

At 12 years of follow-up in the Cancer Survivorship clinic, she continues to do well with no evidence of recurrence and no neurologic sequelae of this lesion or its treatment. A repeat head MRI was performed in 2014, revealing remnant bony damage to the skull and scar tissue but no sign of recurrence (Fig. 2).

3. DISCUSSION

Bony metastatic disease in WT with favorable histology or diffuse anaplasia is uncommon; solitary bone metastases are exceedingly rare. In a pathologic review of metastatic disease in 1396 patients, Marsden, *et al.* identified 18 patients (1.3%) with bony metastatic disease. Only three patients in this cohort had solitary bone metastases (0.2%).[2] Bony metastasis in WT is associated with poor outcomes.[3] There are few reports of metastatic disease to the cranium. Fratkin, *et al.* reported on a 2-year-old boy presenting with an orbital metastatic focus a year after treatment; this patient also had multiple extracranial bony lesions and a

local recurrence in the pelvis. He succumbed to disease at the age of 3 years.[4] Bond, *et al.* published the case of a 14-month-old boy presenting with a lump over his right ear and a palpable abdominal mass. He ultimately developed widespread metastases and died eight months after nephrectomy.[5] To our knowledge, there has not been a previous report of either a solitary metastatic lesion to the skull or long-term survival after metastatic disease to the skull.

4. CONCLUSION

Here we present the previously unreported clinical scenario of the successful treatment of a 3-year old girl presenting with a solitary skull metastatic lesion following initial treatment of localized WT with diffuse anaplasia.

References

1. Breslow NE, Churchill G, Nesmith B, et al. Clinicopathologic features and prognosis for Wilms' tumor patients with metastases at diagnosis. Cancer. 1986; 58(11):2501–2511. [PubMed: 3021319]
2. Marsden HB, Lennox EL, Lawler W, Kinnier-Wilson LM. Bone metastases in childhood renal tumours. British journal of cancer. 1980; 41(6):875–879. [PubMed: 6252920]
3. Watanabe R, Takahashi A, Suzuki M, et al. Adolescent wilms tumor with intraspinal and bone metastases: a case report and the review of literature. Journal of pediatric hematology/oncology. 2009; 31(1):45–48. [PubMed: 19125087]
4. Fratkin JD, Purcell JJ, Krachmer JH, Taylor JC. Wilms' tumor metastatic to the orbit. Jama. 1977; 238(17):1841–1842. [PubMed: 198592]
5. Bond JV, Martin EC. Bone metastases in Wilms' tumour. Clinical radiology. 1975; 26(1):103–106. [PubMed: 164315]

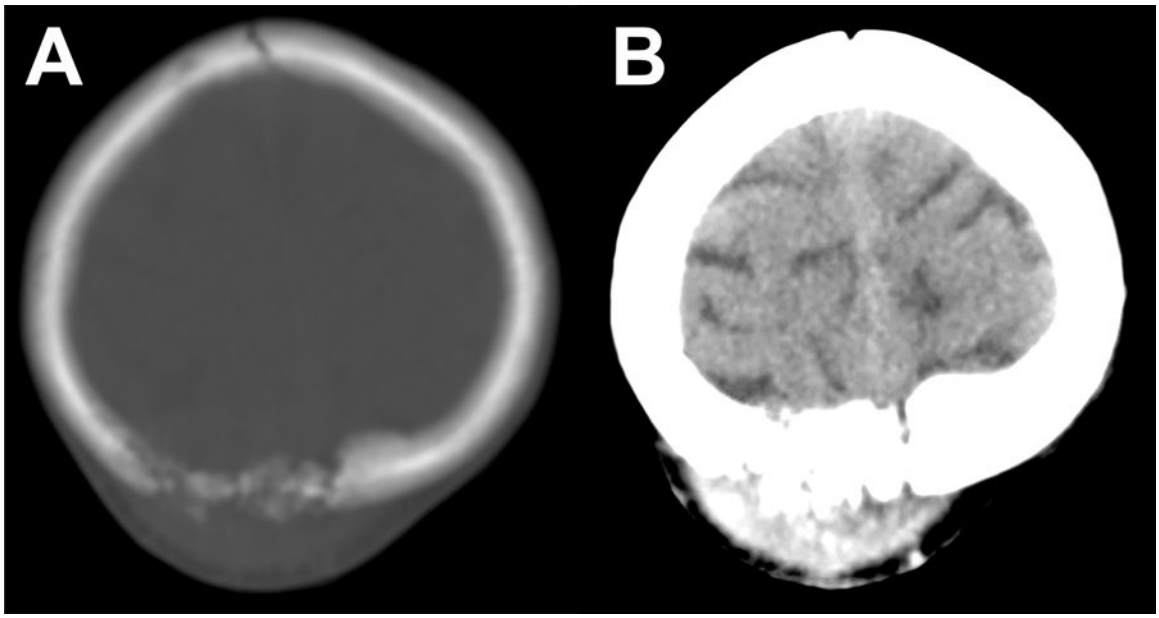

Figure 1.
Head CT scan showing tumor invading the skull and extending to the epidural space and the subgaleal plane (1A, bony window, 1B, soft tissue window).

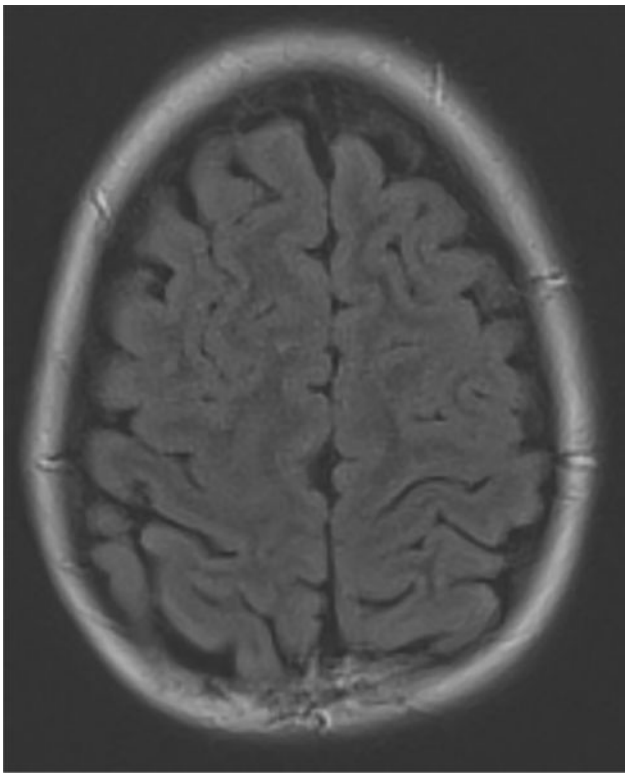

Figure 2.
Head MRI at 14 years of age showing persistent destruction of bone without evidence of recurrence.